Is subcortical vision necessarily mediated by the superior colliculus?

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Perhaps Campion, Latto & Smith are unable to obtain artifact-free evidence for subcortically mediated residual vision in humans because their behavioural methods, like those of the others they cite, are biased against the known properties of the subcortical visual system. Human residual vision has been studied on the assumption that its substrate is the superior colliculus; yet there is now evidence indicating that other brainstem visual centres are just as important – or more so – for both normal and residual vision in nonhuman species. Since these structures mediate capacities much different from those identified with the superior colliculus, incorporating them in our model of the visual system leads to different expectations of residual vision.

Strict application of the "two visual system" hypothesis requires that cortical and subcortical visual mechanisms possess complementary functions and that these be fundamentally the same in all species. In other words, the visual capacities lost after subcortical injuries should match those retained after striate cortex removal, and these should be the same in all species possessing homologues of striate cortex and the pertinent subcortical centres.

In the hooded rat, destruction of the superior colliculus produces no detectable effect on either form or visual intensity discrimination when conventional procedures are used (Legg & Cowey 1977b). Only when stimulus/response compatibility is low do discrimination impairments emerge (Milner, Goodale & Morton 1979). Imapirments in orienting and defensive reactions have also been described (Goodale & Murison 1975), but these occur only with certain stimulus configurations (Goodale, Foreman & Milner 1978; Midgley & Tees 1981). In contrast, lesions involving the ventral lateral geniculate nucleus (LGv) reliably impair visual intensity discrimination performance without affecting line orientation discrimination (Horel 1968; Legg & Cowey 1977a; 1977b). This dissociation is important because visual cortex ablation produces entirely the opposite effect (Horel, Bettinger, Royce & Meyer 1966).

Lesions involving the pretectum have also been found to produce intensity discrimination impairments but these are not as severe as those obtained with LGv lesions (Legg & Cowey 1977a) or only appear when contour information has been eliminated (Blochert, Ferrier & Cooper 1976). The siting of pretectal lesions is crucial since if they extend into the adjacent medial thalamus a severe impairment in line orientation discrimination appears (Legg 1977).

Recent work suggests that these discrimination impairments have a distinct sensory basis. Following the suggestion that cortical and subcortical mechanisms have different spatial frequency sensitivity characteristics (Dean 1978; Legg & Cowey, 1977a), we (Legg & Turkish 1982) assessed the effects of brainstem lesions on spatial contrast sensitivity, but with disappointing results (Legg & Turkish 1982). Lesions involving the medial posterior thalamus and rostral pretectum severely depressed contrast sensitivity, as might have been expected from the line orientation discrimination impairment previously found with such lesions (Legg 1977), but neither LGv lesions nor those restricted to the pretectum produced the expected low spatial frequency loss. However, when flicker sensitivity was measured, LGv lesions were found to produce a marked impairment in detecting low frequency sinusoidal flicker when the stimulus was a uniform field measuring 24° by 20° (Legg 1981). A similar impairment emerged after pretectal lesions but, as with the suprathreshold discrimination work, superior colliculus lesions were without measurable effect. Knowledge of whether or not the effects obtained with LGv or pretectal lesions truly complement those of visual cortex ablation awaits the outcome of studies currently being conducted.

While these observations do not preclude a role for the superior colliculus in residual vision they do indicate that other systems may be at least as important. If these other systems are also available in humans it must be asked why it is not possible to obtain artifact-free evidence of subcortically mediated light detection at least. The answer most probably lies in the stimuli used. In the studies described above the stimuli were large, in contrast to the small spots of light conventionally used in perimetry. The flicker sensitivity measurements also indicate that the systems are tuned to slow stimulus changes; perimetry studies conventionally use brief flashes. From what is known of the single cell response properties of both the pretectum (Sprague, Berlucchi & Rizzolatti 1973) and the LGv (Hale & Sefton 1978) there is a bias towards large, tonic stimuli and uniform fields which would render these systems profoundly insensitive to very brief, localised flashes.

If we take as our starting point the empirically determined properties of subcortical visual mechanisms we arrive at very different expectations for human residual vision. While localisation tasks may be relevant to tectal function they are certainly not going to tap the functions of other subcortical sites. Furthermore, the perimetric techniques forced upon researchers by the need to work within limited scotomata make it unlikely that the operation of these other subcortical mechanisms will be demonstrated directly using conventional methods. Therefore, the results of Campion et al. are entirely in accord with *a* "two visual system" hypothesis, although not the specific version currently in vogue. Far from calling into question the validity of extrapolation from nonhuman to human brain and behaviour relationships, as the authors imply, the fact that behaviours believed to be based upon the superior colliculus cannot be demonstrated in cortically blind humans can just as well be seen as a stimulus for a more critical evaluation of its role in residual vision in other species.

How can striate vision contribute to the detection of objects within a homonymous visual field defect?

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In their target article on blindsight, Campion, Latto & Smith conclude from a thorough review of the literature and from their own experiments that all of the phenomena of blindsight hitherto described in patients could also be explained either by light scatter into unimpaired parts of the visual field or by residual vision attributable to spared striate cortex. We do not wish to get involved in a discussion about whether or not extrastriate visual functions can be present in visual field defects of patients with lesions of the striate cortex. We can, however, support Campion et al.'s view that previous discussions of "blindsight" were biased toward the concept of "extrastriate vision," so that the impression could arise that such vision was the only possible reason some patients with homonymous visual field defects were surprisingly little disabled by their visual loss. Since in the vast majority of cases investigated for blindsight only one-fourth to one-half of the visual field was defective (see Table 1 in the target article), the possible contribution of remaining striate vision to visuospatial orientation should at least also be considered.

We accordingly discuss here some ways striate vision might contribute to the detection of objects within a visual field defect. For the sake of simplicity we talk about homonymous hemianopias only (Table 1).

It appears simple to state that, to begin with, *residual striate*

Table 1 (Meienberg). . *How striate vision might help in detecting objects on the side of a homonymous hemianopia*

1. *Eyes fixating:*	*1.1. Residual striate vision within the blind hemifield*
	1.1.1. Normal striate vision (e.g. spared temporal crescent)
	1.1.2. degraded striate vision (e.g. Riddoch (1917) phenomenon)
	1.2. Cues from the sighted hemifield to objects in the blind hemifield
	1.2.1. Scattered light
	1.2.2. "Incomplete" objects
2. *Eyes moving:*	*Scanning of the side of the blind hemifield with the sighted hemifield* using oculomotor searching strategies

vision within the blind hemifield should be taken into consideration. However, one has to remember that, not infrequently, homonymous hemianopias attributable to lesions of the cerebral hemispheres fail to be congruent, especially in the periphery (Körner & Teuber 1973). A small area of preserved striate vision in the eye with the temporal visual field defect can markedly improve visuospatial orientation (Benton, Levy & Swash 1980; Meienberg 1981). Furthermore, some degraded striate vision consisting of movement and light perception only, as described by Riddoch (1917) or Zappia, Enoch, Stamper, Winkleman & Gay (1971), can still be present within a hemianopic field. The difficulties of differentiating degraded striate vision from "extrastriate" vision have been discussed by Campion et al. They have also investigated a further factor that might help to detect objects within a blind hemifield, namely, scattered light. But not only light scatter can *give the sighted hemifield cues to objects in the blind hemifield.* Parts of objects perceived on the seeing side also point at something else on the blind side. The significance of "incomplete" objects seen in one hemifield is, in our opinion, not the eventual induction of a so-called completion phenomenon, which has already been shown to be an illusion (Gassel & Williams 1963). Such objects do, however, constitute strong stimuli for a patient to move his eyes toward the side of his hemianopia in order to see the objects fully. In a previous study we documented and defined *oculomotor searching strategies* that patients with a homonymous hemianopia employ to compensate for their visual loss (Meienberg, Zangemeister, Rosenberg, Hoyt & Stark 1981). Further recordings of eye movements in hemianopic patients confirm our first impression that most of them improve their search strategies with time; that is, they learn to scan space optimally with the remaining half of their visual field, and thus minimize the chance of missing objects appearing on their blind side. Even without any object for fixation, in front of a featureless gray screen, patients with good oculomotor compensation frequently make large searching saccades toward the side of the blind hemifield (Harrer & Meienberg, unpublished data).

We do not wish to imply that in all "blindsight" studies factors like those mentioned above have been ignored. We merely wish to call attention to possible reasons for relatively little disability from a homonymous hemianopia other than extrastriate vision. Even if some primitive visual functions of extrastriate origin in a hemianopic field exist, it remains unclear whether a patient would use them or whether he would prefer instead to try to catch as much visual information as possible with his remaining and normally functioning visual field areas.

From what has been said above, extrastriate vision research should focus more on patients with complete blindness from destruction of both striate cortices. Until now only a few such patients have been examined for extrastriate visual functions, and the findings are in part contradictory (Bender & Krieger 1951; Brindley, Gautier-Smith & Lewin 1969; Celesia, Archer, Koroiwa & Goldfader 1980; Perenin, Ruel & Hécaen 1980). Since in several patients with cortical blindness almost normal flash-evoked potentials could be recorded (Bodis-Wollner, Atkin, Raab & Wolkstein 1977; Celesia et al. 1980; Hess, Meienberg & Ludin 1982; Spehlmann, Gross, Ho, Leestma & Norcross 1977), a substrate for extrastriate vision also seems to exist in man.

Scotomas and the visual field

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Campion, Latto & Smith (henceforth "the authors") are not just unconvinced by the data on blindsight, they are not sure what claim it is meant to support. If we define blindsight as seeing without being aware of seeing, then its existence is neither surprising nor very interesting. So that cannot be what Weiskrantz and others have been after. The authors therefore define blindsight as vision that is not mediated by the standard pathway to the striate cortex. This does make sense of some, at any rate, of the claims under discussion. It could happen, though, were we to adopt this definition, that subjects were aware of seeing stimuli by blindsight. It wouldn't be very blind. In fact, something like this may be happening with some of the subjects, who describe their experiences as not like seeing but not, once they had become used to the situation, as something of which they were completely unaware.

Which terms one has to be careful about depends on which others one takes for granted. An illustration of this is the very term "scotoma." If locations in the visual field could be simply correlated both with specific points in the cortex and with conscious reports of stimulation at those points, then all would be easy. But such simple correlations do not exist, and we did not need work on blindsight to tell us that. Yet we persist in our picture of a scotoma as a blank patch in the visual field, though we know it may matter whether we take this blankness as lack of awareness or lack of response or lack of response to a particular class of stimuli. (Signal detection theory brings out clearly some of the ways in which these may differ.)

There are two crucial propositions here. If they were both true we would not have to be so careful about what we build into our terms.

a. Subjects' unforced reports of what they see correlate, in terms of form and location, with their capacities to respond; any differences will be of degree, depending on thresholds and the like.

b. Pattern recognition and location of stimuli in visual space are based on connections with the striate cortex.

It follows from these that

c. Conscious (verbal) responses to visual stimuli are based on connections with the striate cortex.

If (a) is true and blindsight data are taken at face value, then (b) is false. If (c) is true then the possibilities I described in my first paragraph cannot obtain. The authors suggest that we should not rest very much on (a). They offer ways of explaining the data that preserve (b). Two of these, the use of scattered light and the presence of residual striate capacity, actually cast doubt on (a), for it is clear that there are scotoma here, in the sense of regions of the subjects' field of view of which they can report no impression, but that subjects can respond to stimuli from these regions. In forced-choice situations subjects can even describe qualities of these regions, but will not describe themselves as being *aware* of them. (That is, they would not mention them if asked to report on what they see; see Dennett 1978b.)

It seems clear, then, that what is really under attack here is